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FRONTAL LESIONS AND BEHAVIORAL INSTABILITY[1]

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Fine grain analysis showed that frontal ablation lowered the level of visual discrimination performance and augmented fluctuations in behavior after criterion had been met. These results are consonant with earlier ones and suggest that one important consequence of frontal lesion in the monkey is the production of behavioral instability.

The functions of the frontal eugranular cortex continue to puzzle neurobehavioral scientists (Warren & Akert, 1964). Much of the effective analysis of the problem has stemmed from the observation of Jacobsen (1936) that ablations of this part of the forebrain interfere with a monkey's ability to solve the delayed response problem. Attention has been focused on this observation since ablations of other parts of the cerebral cortex do not impair the solution of this task (Pribram, 1954). Such frontal ablations have, on the whole, little effect on the performance of simple visual discriminations (Pribram, Mishkin, Rosvold, & Kaplan, 1952); yet, when more complicated discrimination procedures are used, frontally lesioned monkeys do show differences from normal *Ss* in the way these problems are handled (Harlow, 1949; Pribram, 1960).

Recently, procedures of testing and of data analysis have been refined. Automated testing by computer-programmed devices and mathematical analysis of trial-by-trial change, in performance are now available. It was felt, therefore, that another look at the way in which frontally ablated monkeys learn and perform on a simple, simultaneously presented discrimination would prove fruitful.

METHOD

Eight experimentally naive rhesus monkeys were tested in an automated discrimination apparatus that allows discrete trial analysis (DADTA) (Pribram, Gardner, Pressman, & Bagshaw, 1962). Briefly, this apparatus consists of 16 microswitch panels arranged in four rows of four, each with the capacity of displaying any one of 12 different visual patterns. The apparatus is programmed to deliver a peanut reward through a centrally placed feeder box whenever a certain pattern is responded to by pressing the appropriate panel. The location of a pattern among the 16 panels is scrambled from trial to trial, according to a preset schedule.

All *Ss* were trained to respond to a single red circle, presented at various locations in sequence at 5-sec. intervals. After reaching consistent performance, four *Ss* underwent single-stage bilateral lobectomies, the cortical removal extending from arcuate sulcus to and including the frontal pole. Surgical procedure and histological verification of the lesions were accomplished according to techniques repeatedly presented (Sherer & Pribram, 1962).

After recovery, *Ss* were given the discrimination task; the numerals 4 and 6 served as discriminanda. As before, cue positions on the DADTA screen were scrambled from trial to trial. The correct contingency was balanced among *Ss*, half receiving consistent rewards on 4 and half on 6. Fifty trials per day were presented at 5-sec. intervals until an arbitrary criterion of 43 correct choices out of any 50 consecutive trials was reached. Following an interval of approximately 1 wk., another 50 trials on each of 5 days were given under the same test conditions.

RESULTS

Discrimination Learning

The control (unoperated) group achieved criterion in an average of 177 trials (individual scores of 90, 140, 140, and 340). The frontally lesioned group required an average of 275 trials (individual scores of 150, 250, 250, and 450). For these two distributions of scores the Mann-Whitney U of 3 generates a two-tailed p of .200. Presented in Figure 1 are the backward learning curves for control and frontal *Ss*.

Discrimination Performance

The results of the posteriterion performance run are also shown in Figure 1. In this series of 250 trials the number of correct trials for controls were 242, 243, 247, and 250, and for frontals, 198, 224, 225, and 231. The mean numbers of correct trials were, respectively, 245.5 (98.5%) and 219.5 (88%). Note that there was no overlap in the distributions of posteriterion performance scores. The Mann-Whitney U of 0 generates a two-tailed p of .028.

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The plots of individual performance protocols in the posteriterion period suggested that frontal *Ss* were more variable than controls. As a measure of variability we adopted the number of performance dips per block of 10 trials. More specifically, we considered each point of an individual *S*'s protocol (pre- or posteriterion) as plotted in the format of Figure 1, i.e., percentage correct over 10-trial blocks, and then asked if the next succeeding point showed a performance dip (i.e, was lower). These dips or performance lapses were totaled and divided by the number of 10-trial blocks over which the lapses were observed. The preciterion period extended from the criterion point back 90 trials (the last point including all eight *Ss*). The posteriterion point extended from the criterion point forward 250 trials.

Mean performance lapses for control and frontal *Ss* in the preciterion period were about the same (see Table 1). In the posteriterion period, however, control *S*'s performance was considerably less variable than frontal *S*'s performance. For the two posteriterion distributions of scores, the Mann-Whitney U of 1 generates a two-tailed p of .058. A t test on the same data gives a p value $> .02$ but $< .05$ ($t = 2.75$, $df = 7$).

Discussion

The results of this experiment hint at a very slight retardation of discrimination learning by frontally lesioned monkeys. Much more striking, however, is their impaired posteriterion performance. This impairment is characterized by a lower mean performance and by considerable irregularity in behavior when 10-trial blocks are compared. The experiment does not allow us to distinguish whether these fluctations in behavior lead to or are a consequence of the lower level of performance. However, on the basis of earlier experiments (Pribram, Ahumada, Hartog, & Roos, 1964) which showed that frontally lesioned monkeys behave more randomly in a variety of situations, the current results can be taken to support the hypothesis that irregularities in behavior are an important consequence of frontal lesion in primates.

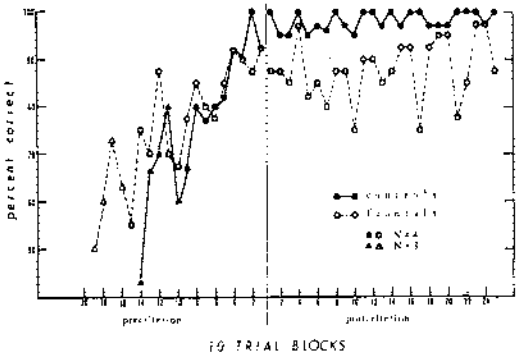

Fig. 1. Mean percentage of correct responses by trial blocks of 10 for control and frontal *Ss*.

TABLE 1

Performance Lapse Scores for Control and Frontal *Ss* in Pre- and Posteriterion Periods

	Preciterion scores		Posteriterion scores	
	Controls	Frontals	Controls	Frontals
	.22	.22	.24	.24
	.22	.44	.12	.28
	.33	.33	.12	.48
	.22	.22	.00	.28
M	.25	.30	.12	.32

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